NUTRITION SUPPORT IN ACUTE KIDNEY INJURY

BY

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Lect. Of internal medicine

Presentation overview

- Epidemiological aspects and pathogenesis of protein-energy wasting (PEW) in AKI
- Nutrient needs in patients with AKI
- Integration between nutrition and renal replacement therapy (RRT) in patients with AKI

Epidemiology and prognosis of AKI

THREE MAJOR PROBLEMS:

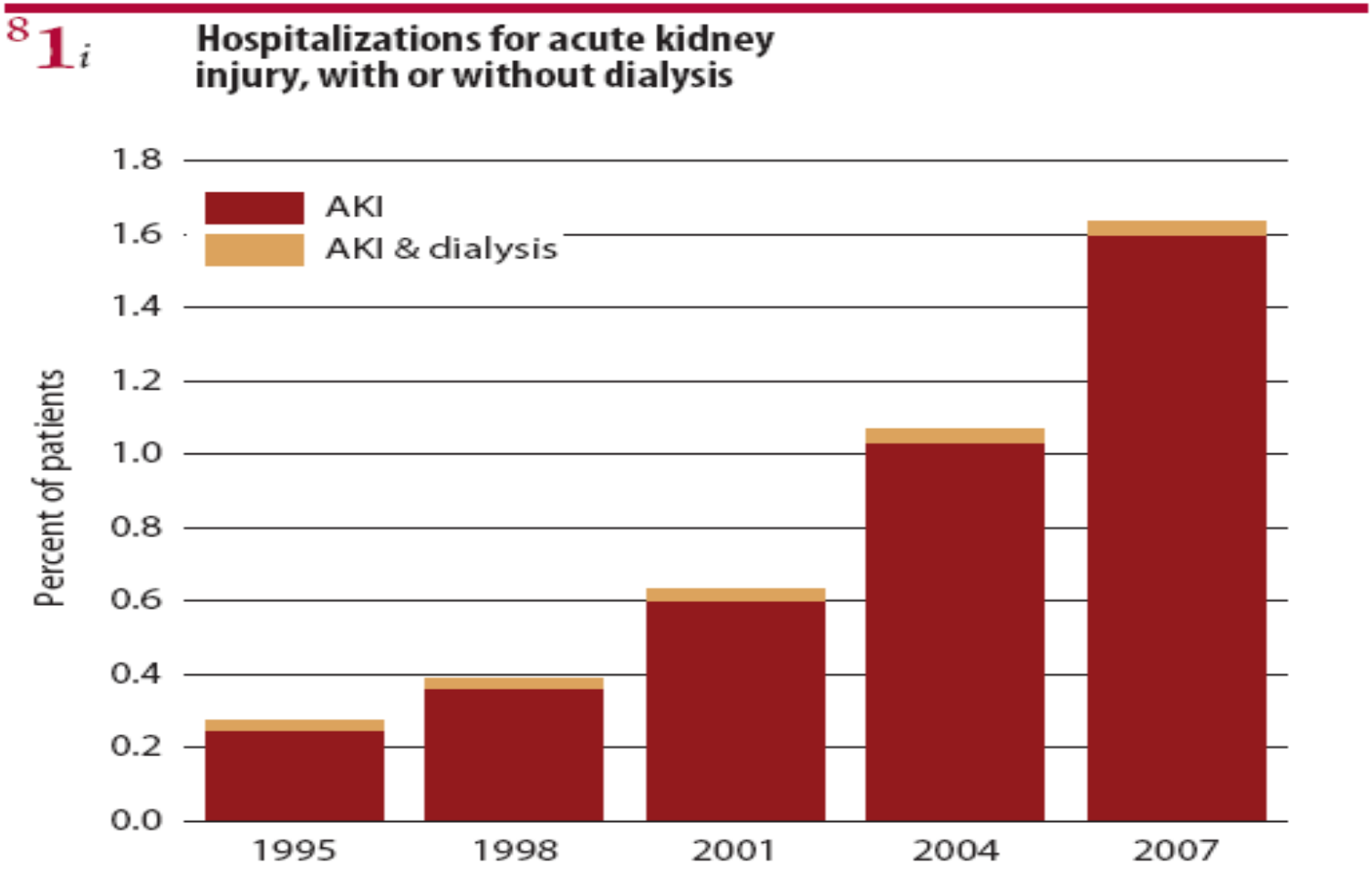
- Incidence is high
- Incidence is increasing
- Negative impact on short- and long-term outcome

World incidence of AKI : meta_analysis june 2013

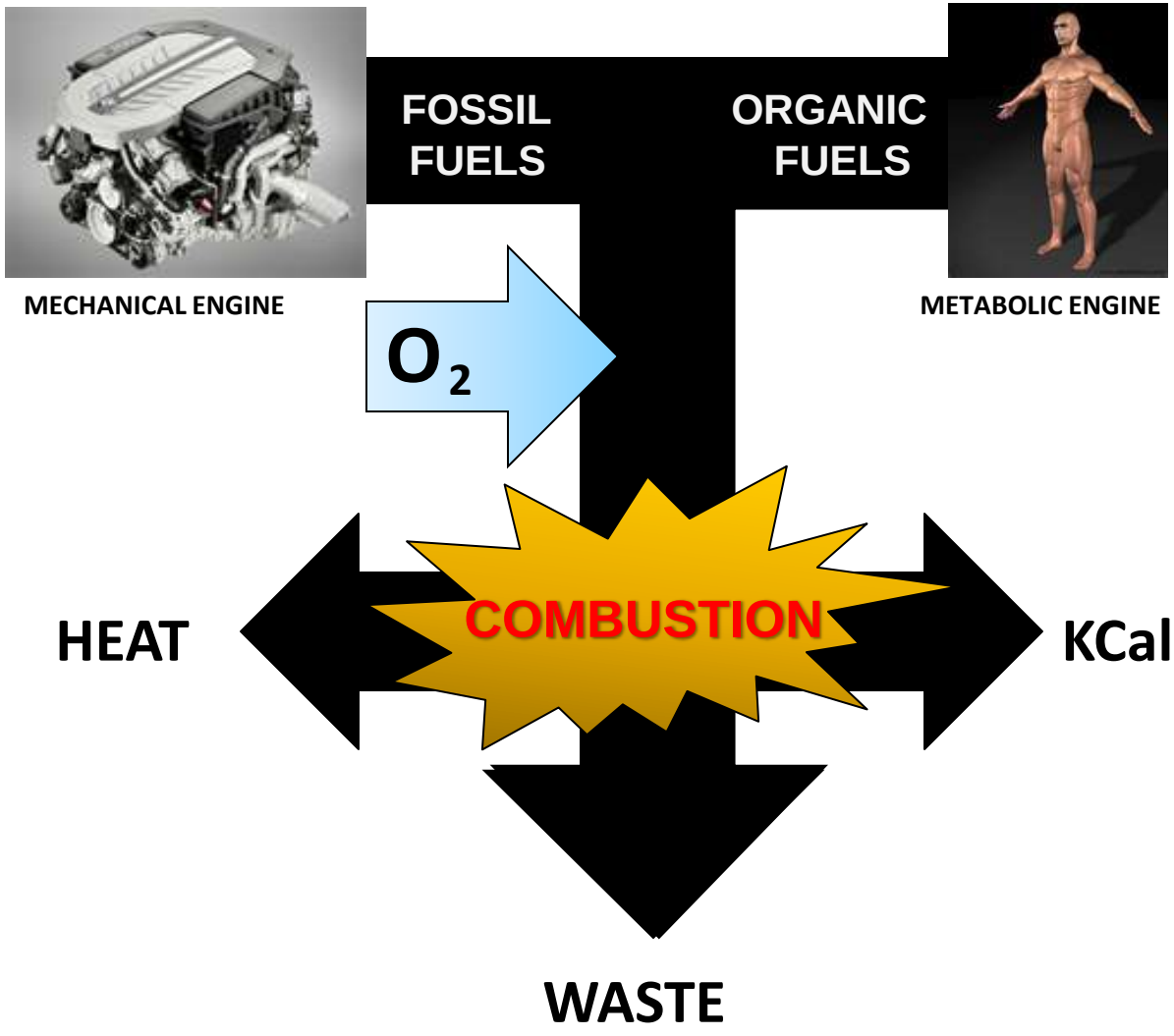
Table 2. Pooled incidence rate of AKI according to the KDIGO-equivalent definition

Subgroup	Studies (*n*)	Patients (*n*)	Patients with AKI (*n*)	AKI Incidence Rate (%)	95% Confidence Interval	Test for Heterogeneity	
						I^2 Index	*Q* Test *P* Value
All	154	3,585,911	573,424	23.2	21.0 to 25.7	99.9	<0.001
Age category							
Adults	130	3,571,691	569,861	21.6	19.3 to 24.1	99.9	<0.001
Children	24	14,220	3563	33.7	26.9 to 41.3	98.3	<0.001
Clinical setting							
Community acquired	7	548,398	4897	8.3	1.6 to 33.0	99.9	<0.001
Critical care	41	888,604	272,580	31.7	28.6 to 35.0	99.9	<0.001
Cardiac surgery	42	164,333	33,157	24.3	20.4 to 28.8	99.7	<0.001
Trauma	4	14,947	2557	19.9	13.6 to 28.2	98.7	<0.001
Heart failure	1	682	221	32.4	29.0 to 36.0	—	—
Hematology/oncology	3	2401	453	21.3	7.5 to 47.6	99.2	<0.001
Nephrotoxins	4	17,786	1681	12.2	6.2 to 22.7	98.7	<0.001
Hospital acquired, unspecified	52	1,948,760	257,878	20.9	17.2 to 25.2	99.9	<0.001

Annual data report 2009



Review of Nutritional Requirements



ORGANIC FUELS

OXIDATIVE METABOLISM OF ORGANIC FUELS	
FUEL	**ENERGY YIELD**
LIPID	**9.1 kcal/g**
PROTEIN	**4.0 kcal/g**
GLUCOSE	**3.75 kcal/g**

DAILY ENERGY EXPENDITURE

- **Predictive equations**
  - BEE (Basal Energy Expenditure) kcal/24 hr
    - Men = 66 + (13.7 x wt) + (5.0 x ht) – (6.7 x Age)
    - Women = 655 + (9.6 x wt) + (1.8 x ht) – (4.7 x age)
  - REE (Resting Energy Expenditure)
    - REE = 1.2 x BEE
- Simplified computations: 20-40 kcal/kg ideal body wt depending on activity
  - Caloric requirements: 70% from carbohydrates & 30% from fats
  - Protein requirements: 0.8 to 1.2 in normal metabolism, 1.2 to 1.8 in hypercatabolism

FUEL STORES IN HEALTHY ADULTS		
FUEL SOURCE	**AMOUNT (KG)**	**ENERGY YIELD (KCAL)**
ADIPOSE TISSUE	15.0	141,000
MUSCLE PROTEIN	6.0	24,000
TOTAL GLYCOGEN	0.09	900
TOTAL KCAL		165,900

METABOLIC ALTERATIONS IN AKI

- **HYPERMETABOLIC STATE**
  - Energy expenditure (EE) being proportional to the amount of stress
  - Although active solute transport in a functioning kidney is an energy-consuming process, the presence of AKI by itself (in the absence of critical illness) does not seem to affect resting EE (REE)
  - EE in AKI patients is therefore determined mainly by the underlying condition. Studies in chronic kidney disease yield conflicting results varying between increased, normal, or even decreased REE.

METABOLIC ALTERATIONS IN AKI

- **'DIABETES OF STRESS'**
  - hyperglycemia and insulin resistance
  - Hepatic gluconeogenesis (from amino acids and lactate) increases mainly due to the action of catabolic hormones such as glucagon, epinephrine, and cortisol
  - The normal suppressive action of exogenous glucose and insulin on hepatic gluconeogenesis is decreased
  - Peripheral glucose utilization in insulin-dependent tissues (muscle and fat) is also decreased
  - In normal conditions, the kidney plays an important role in glucose homeostasis
  - The loss of kidney function by itself may contribute to the altered carbohydrate metabolism in AKI

METABOLIC ALTERATIONS IN AKI

- **PROTEIN CATABOLISM AND NET NEGATIVE NITROGEN BALANCE**
  - The increased protein synthesis is unable to compensate for the higher proteolysis
  - In the acute phase, this catabolic response may be beneficial, providing amino acids for hepatic gluconeogenesis (supplying substrate for vital tissues such as the brain and immune cells) and for synthesis of proteins involved in immune function and in the acute-phase response.

- However, the sustained hyper catabolism in the chronic phase of critical illness results in a substantial loss of lean body mass and in muscle weakness and decreased immune function

- Protein catabolic rates may go up to 1.3 and 1.8 g/kg per day

- Protein catabolism also accelerates the increases of serum potassium and phosphorus

Nitrogen Balance in ARF

- Standard nitrogen balance studies require a creatinine clearance of more than 50 mL/min/1.73m2
- In ARF, urea nitrogen appearance (UNA) is a better method of determining nitrogen balance
- UNA = UUN + change in the urea nitrogen pool

Calculation of Urea Nitrogen Appearance (UNA)

UNA (g) = UUN + [BUN2 – BUN1) x .6 x BW1] + [(BW2-BW1) x BUN2]

Net protein breakdown = UNA x 6.25

UUN = urinary urea nitrogen (g/24hr)

BUN1 = initial collection of blood urea nitrogen, postdialysis (g/L)

BUN2 = final collection of blood urea nitrogen, predialysis (g/L)

BW1 = postdialysis wt (kg)

BW2 = predialysis wt (kg)

METABOLIC ALTERATIONS IN AKI

- **ABNORMAL NUTRIENT PROCESSING**
  - The malnutrition of starvation is due deficits in essential nutrients and nutrient intake will correct the malnutrition
  - The malnutrition in AKI and other critical illnesses is due to a disease-induced abnormal nutrient processing. Nutrient intake alone may not correct the malnutrition. The underlying disease must be addressed

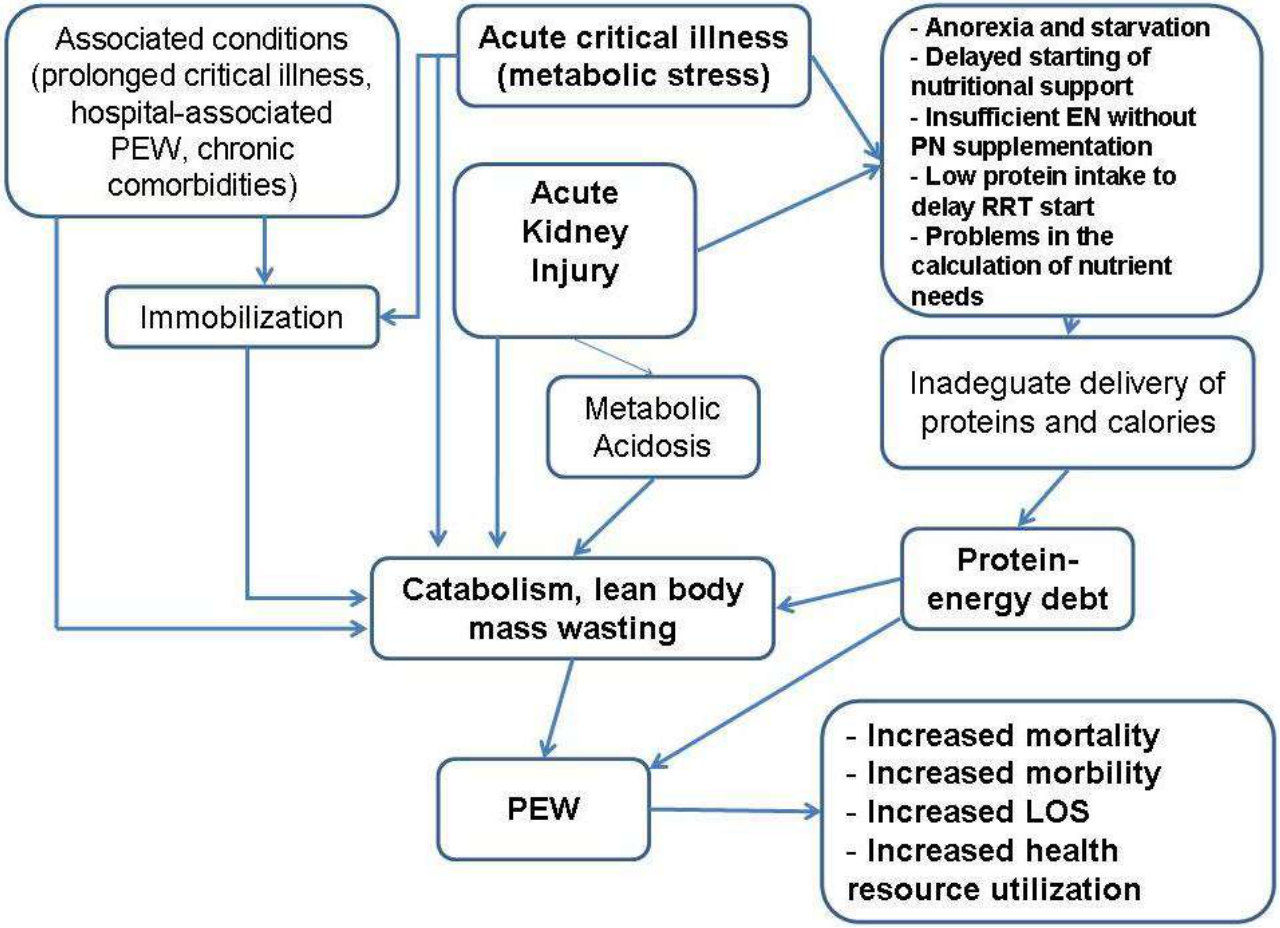
METABOLIC ALTERATIONS IN AKI

- **NUTRIENT TOXICITY**
  - In healthy subjects ,5% of glucose is metabolized to lactate. In critically ill patients, this may rise up to 85%

Specific (and contrasting) nutritional problems in patients with AKI on RRT

- Risk of underfeeding
- Excess (hidden) calories from anticoagulant and replacement fluids for RRT

Pathogenesis of PEW in AKI: underfeeding



Clin. Nutr. Metab Care2013

Mechanism of overfeeding in AKI

- Problems of evaluation of ref. BW
- Glucose in TPN
- Citrate as anticoagulant ,lactate as buffer in RRT
- hyperglycemia, imbalaced electrolytes, fluid over load

- Leading to: worsen morbidity , mortality and restoration of renal function

Nutrition in AKI is a difficult task:

- Dysmetabolism of critical illness worsened by the acute loss of kidney homeostatic function
- Nutritional approach made difficult by the complexity of the syndrome itself and by the frequent need of renal replacement therapy (RRT)
- No data from RCTs
- Clinical practice mostly based on expert's opinions

No major difference as regard ESPEN guidline 2006:2009

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A.S.P.E.N. Clinical Guidelines: Nutrition Support in Adult Acute and Chronic Renal Failure

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ESPEN Guidelines on Parenteral Nutrition: Adult Renal Failure

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A major open problem in AKI is the lack of adequate tools for nutritional ass.

TABLE 1. Nutritional status evaluation variables in acute kidney injury (AKI) and their limitations

Nutritional parameters	Limitations
Albumin, prealbumin, cholesterol	They can be reduced even independently from PEW (negative markers of inflammation)
Lymphocyte count	Low specificity
Body weight (BW) changes	Total body water increased in AKI Fluid overload can mask lean body mass changes
Anthropometry (triceps skinfold, midarm circumference, etc.)	Interference by arm edema
Protein catabolic rate (PCR) or protein equivalent of nitrogen appearance (PNA)	Measurement requires calculations based on urea kinetic during RRT + dialysis fluid collection/proportional sampling
Energy expenditure (EE)	Formulas for EE prediction not always reliable in critically ill patients (often based on body weight) Indirect calorimetry not always available in the ICU or nephrology wards
Multidimensional nutritional scoring systems (SGA and its modifications)	Most data are from chronic renal failure patients
Potential tools or in development	
Laboratory markers	
Growth hormone and IGF-1 levels	Few data available in AKI
Inflammatory markers (PCR, serum interleukine levels, etc)	Markers of patient' outcome/risk for PEW; not nutritional parameters (not useful for nutritional diagnosis or monitoring)
Body mass and composition	
Total body nitrogen	Research tools (cumbersome and/or costly and/or invasive)
Energy-beam-based methods	Research tools (cumbersome and/or costly and/or invasive)
Muscle fiber size and composition	Research tools (cumbersome and/or costly and/or invasive)
Bioimpedence analysis	No data in AKI
CT and/or MRI	No data in AKI

PEW, protein-energy wasting; RRT, renal replacement therapy; SGA, Subjective Global Assessment.

Who Should Get Nutritional Support?

Patients who:

- Cannot meet nutrient requirements
- Have documented inadequate oral intake
- Have unpredictable return of GI function
- Need a prolonged period of NPO/bowel rest

NUTRITIONAL SUPPORT

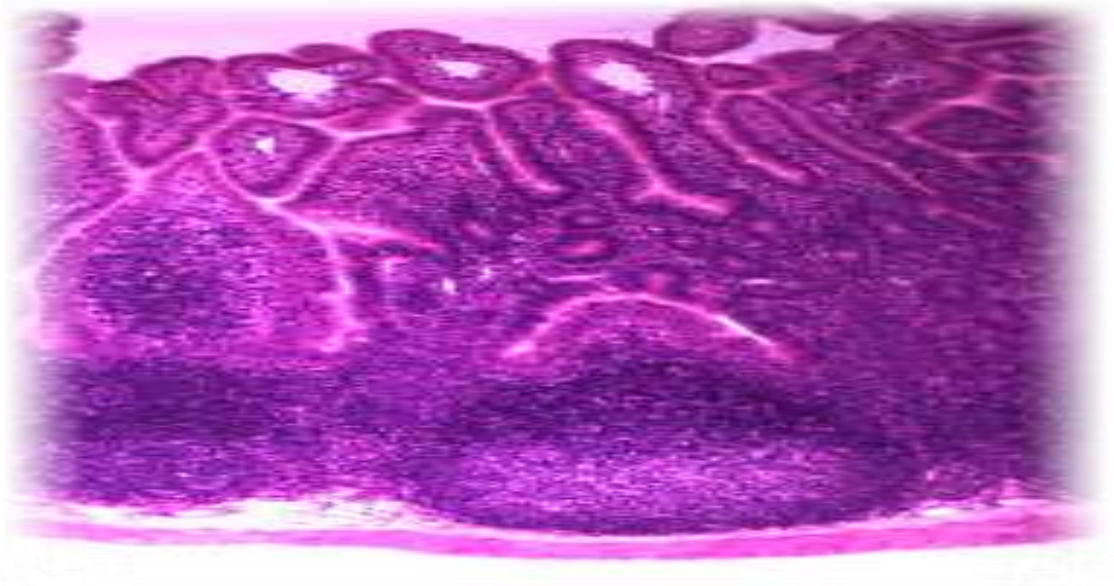
- **ENTERAL NUTRITION (EN) IS ALWAYS BETTER THAN PARENTERAL NUTRITION (PN)**
  - Meta-analyses comparing EN with PN - no difference in mortality
  - Lower incidence of infectious complications with EN
    - may be explained by the higher incidence of hyperglycemia in patients receiving PN

IF THE GUT IS AVAILABLE, USE IT!!!

NUTRITIONAL SUPPORT IN AKI

- **EARLY VERSUS LATE EN**
  - Meta-analysis showed **reduced infectious complications** and length of hospital stay with early EN, but no effect on noninfectious complications or mortality
  - However, **enterally fed critically ill patients often do not meet their nutritional targets**, especially in the first days of ICU stay
  - **Adequate early nutrition is easier with the parenteral route**
  - Most of the mortality benefits of PN suggests that **PN should be given when EN cannot be initiated within 24 hours of ICU admission**

The rationale for early EN



- » Use of the gut stimulates GALT & MALT ➡ resulting in enhanced immune response
- » Early feeding can trigger gut immunity and thereby improve outcomes

- Delay or failure may promote a pro-inflammatory state with ⇧ disease severity & morbidity

McClave, J Clin Gastro, Sept 2002

The rationale for early EN

- Absence of gut stimulation is associated with gut atrophy
  - Changes in gut integrity begin w/in 6 hrs
- Higher incidence of infection/ sepsis

Zaloga GP. Crit Care Med 1999;27:259
McClave, J Clin Gastro, Sept 2002

NUTRITIONAL SUPPORT IN AKI

OPTIMAL AMOUNT OF CALORIES

- **Overfeeding should be avoided**
  - Hyperglycemia
  - excess lipid deposition
  - Azotemia
  - excess carbon dioxide (CO_2) production with difficult weaning from the respirator
  - infectious complications

NUTRITIONAL SUPPORT IN AKI

OPTIMAL AMOUNT OF CALORIES

- Although not based on solid evidence, recent recommendations suggest a **non protein energy supply**

  - 25 to 30 kcal/kg per day in men and 20 to 25 kcal/kg per day in women

  - The proposed proportions of nonprotein energy supply are 60% to 70% of carbohydrate and 30% to 40% of fat

NUTRITIONAL SUPPORT IN AKI

PROTEIN INTAKE :

- **Goal is to improve protein synthesis and nitrogen balance**
  - Although negative nitrogen balances are associated with the worst outcomes, there are no randomized studies comparing different protein or nitrogen intakes with regard to clinical outcomes in ICU patients
  - Although the ideal amount is still debated, a protein intake of between **1.2 and 1.6 g/kg per day** (0.16 to 0.24 g nitrogen/kg per day) is usually recommended
  - Because many nonessential amino acids are not readily synthesized or increasingly used in critically ill patients, the **combination of essential and nonessential amino acids is supposed to be superior**

ROLE OF SPECIFIC COMPONENTS

- **Glutamine**
  - **Important fuel for cells of the immune system**
  - **In stress situations, concentrations decrease.**
  - **Available guidelines recommend enteral and parenteral supplementation**
- **Antioxidant micronutrients**
  - Micronutrients (vitamins and trace elements) play a key role in metabolism, immune function, and antioxidant processes, AKI patients have increased oxidative stress
  - They are deficient in critically ill patients and should be supplemented
  - **Selenium, zinc, vitamin E, and vitamin C** show promising effects on infectious complications and/or mortality in ICU patients

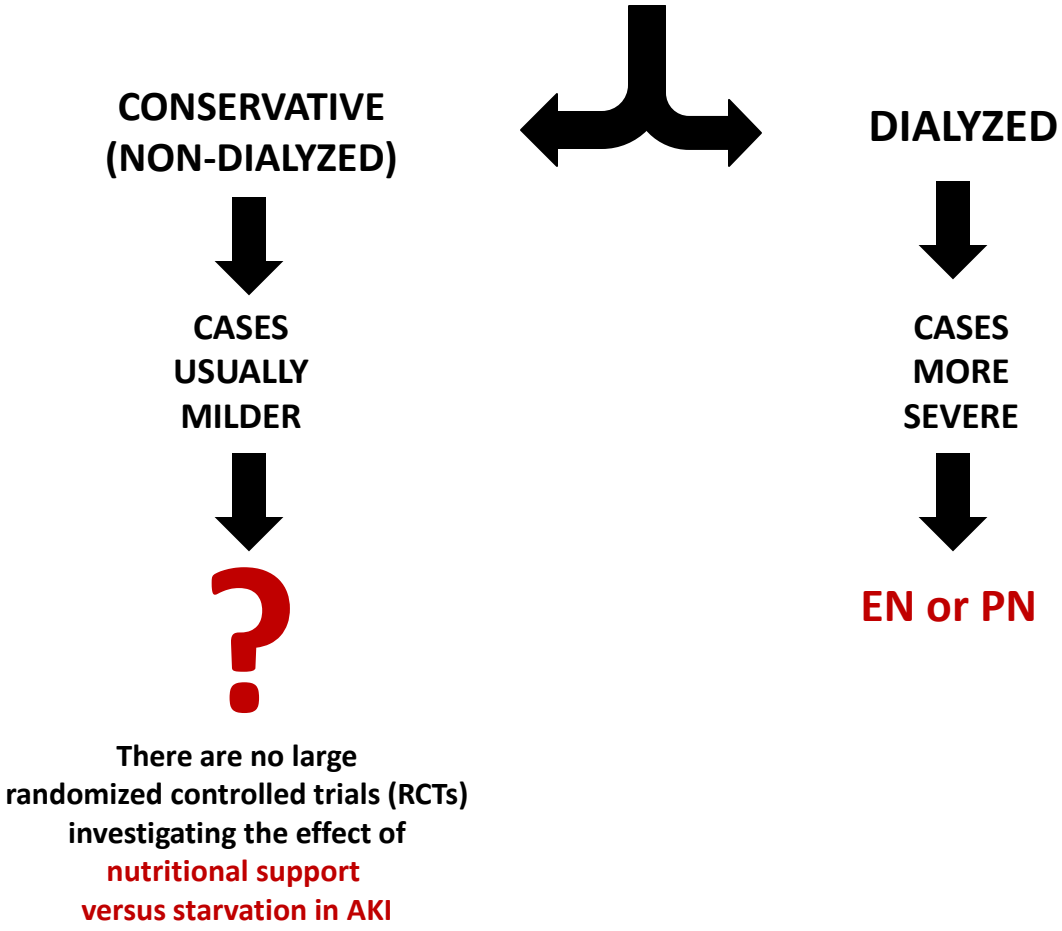
ROLE OF SPECIFIC COMPONENTS

- **Immunonutrients**
  - Nutrients with an immune-modulating effect include: **glutamine, arginine, nucleotides, and omega-3 fatty acids**

    - Meta-analysis aggregating the results of three RCTs of enteral supplementation of omega-3 fatty acids (fish oil) in patients with acute respiratory distress syndrome demonstrated that enteral formula enriched with fish oils significantly reduces mortality and ventilator days and tended to reduce ICU length of stay. A role for exogenous omega-3 fatty acids in human renal protection is, at this moment, purely speculative.

RECOMMENDATIONS FOR NUTRITION IN ACUTE KIDNEY INJURY

ACUTE KIDNEY INJURY



MNT for Adult ARF

- Energy: BEE X 1.2-1.3 or 25-35 kcal/kg
- Protein: .8-1.2 g/kg noncatabolic, without dialysis; 1.2-1.5 g/kg catabolic and/or initiation of dialysis
- Fluid: 24 hour urine output + 500 ml (750-1500 ml)
- Sodium: 2.0-3.0 grams
- Potassium: 2.0-3.0 grams
- Phosphorus: 8-15 mg/kg; may need binders; needs may increase with dialysis, return of kidney function, anabolism

Source: Byham-Gray, Wiesen, eds. A Clinical Guide to Nutrition Care in Kidney Disease. ADA, 2004

Nutritional support for acute kidney injury

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Authors' conclusions

- **There is not enough evidence to support the effectiveness of nutritional support for AKI. Further high quality studies are required to provide reliable evidence of the effect and safety of nutritional support**

Our conclusion

- A)Protein-Energy Wasting is frequent in AKI.
- b)At the present time no definitive demonstration from RCTs is available concerning the positive effects of artificial nutrition on prognosis in AKI patients; however, artificial nutrition should be considered a key component of therapeutic strategy of the syndrome
- c)Energy at not more than 25-30 Kcal/Kg/day and proteins at 1.5-2 g/Kg/day should be provided in critically ill patients with AKI on RRT
- d)Enteral nutrition should be the initial modality for artificial nutrition in AKI; in most cases it should be integrated with PN
- e)More adequate nutritional support with daily RRT


THANK
YOU